WHAT IS CLAIMED IS:

1. A non-natural transgenic mouse with a disruption in at least one allele of the corticotropin releasing factor receptor 2 (CRFR2) such that said mouse does not express corticotropin releasing factor receptor 2 protein from said allele.

2. The transgenic mouse of claim 1, wherein the DNA sequences for exons 10, 11, and 12 of said corticotropin releasing factor receptor 2 allele have been deleted.

3. The transgenic mouse of claim 2, wherein said DNA sequences have been replaced with a neomycin resistance gene cassette.

4. The transgenic mouse of claim 3, wherein said mouse is heterozygous for said replacement.

5. The transgenic mouse of claim 3, wherein said mouse is homozygous for said replacement.

6. The progeny of a mating between a mouse of claim 3 and a mouse of another strain.

7. A method of screening a compound for anxiety modulating activity, comprising the steps of:

a) administering said compound to the transgenic mouse of claim 5;

b) testing said mouse for anxiety-related behavior; and,

c) comparing anxiety-like behavior of said mouse with anxiety-like behavior in a second transgenic mouse of claim 5 to which said compound was not administered.

8. The method of claim 7, wherein said mice are tested for anxiety in an elevated plus maze.

9. A method of screening a compound for depression-modulating activity, comprising the steps of:

a). administering said compound to the transgenic mouse of claim 5;

b). testing said mouse for depression-like behavior; and,

c). comparing depression-like behavior of said mouse with depression-like behavior in a second transgenic mouse of claim 5 to which said compound was not administered.

10. A method of screening for compounds which control blood pressure, comprising the steps of:

a). administering a compound to the transgenic mouse of claim 5;

b). testing said transgenic mouse for alterations in blood pressure; and,

c). comparing alterations of blood pressure in said transgenic mouse with alterations of blood pressure in a second mouse, wherein said second mouse is selected from the group consisting of a transgenic mouse of claim 5 to which said compound

was not administered and a wild type mouse to which said compound was also administered.

11. A method of screening for compounds which affect angiogenesis, comprising the steps of:

a). administering a compound to the transgenic mouse of claim 5;

b). assaying said transgenic mouse for alterations in angiogenesis; and,

c). comparing alterations of angiogenesis in said transgenic mouse with alterations of angiogenesis in mice selected from the group consisting of transgenic mice of claim 5 to which said compound was not administered and wild type mice to which said compound was administered.

12. A method of screening a compound for effects on the response of the hypothalamic-pituitary-adrenal axis to stress, comprising the steps of:

a). administering said compound to the transgenic mouse of claim 5;

b). placing said mouse in a stress-inducing situation,

c). monitoring plasma levels of corticosterone and adrenocorticotropic hormone in said mouse; and,

d). comparing said levels to those in a transgenic mouse of claim 5 not placed in said stress-inducing situation.

13. The method of claim 12, wherein said stress-inducing situation is physical restraint-stress.

14. A method of determining the effects of CRFR2 on a second protein, comprising the steps of

a). administering an agonist that affects the second protein to the transgenic mouse of claim 5;

b) performing an assay of the second protein, wherein said assay is selected from the group consisting of assays of protein expression and assays of protein activity; and,

c). comparing assay results on said transgenic mouse with those obtained from a wild type mouse administered the same agonist.

15. The method of claim 14, wherein said second protein is selected from the group consisting of corticotropin releasing factor, corticotropin releasing factor receptor 1, urocortin, corticotropin receptors and urocortin receptors.

16. A method of stimulating increased angiogenesis in a target tissue comprising the step of administering a CRFR2 antagonist to said target tissue.

17. The method of claim 16, wherein said CRFR2 antagonist is an antisense nucleotide directed against CRFR2.

18. The method of claim 16, wherein said target tissue is selected from the group consisting of heart, brain, pituitary, gonad, kidney, adipose, and gastrointestinal tract tissues.

19. The method of claim 16 wherein said angiogenesis is increased in an individual having a pathophysiological condition selected from the group consisting of infarction, stroke, and injury.

20. A method of inhibiting angiogenesis in a target tissue comprising the step of administering a CRFR2 agonist to said target tissue.

21. The method of claim 20 wherein said CRFR2 agonist is selected from the group consisting of urocortin and CRF.

22. The method of claim 20, wherein said tissue is selected from the group consisting of heart, brain, pituitary, gonad, kidney, adipose, and gastrointestinal tract tissues.

23. The method of claim 20 wherein said angiogenesis is inhibited in an individual having a pathophysiological condition selected from the group consisting of cancer and diabetic retinopathy.

24. A method of stimulating hair growth comprising the step:

contacting urocortin with a region of skin on which hair growth is desired.

25. The method of claim 24, wherein said urocortin is implanted under the skin.

26. The method of claim 24, wherein bFGF is administered to said skin before urocortin, after urocortin or simultaneously with urocortin.

27. The method of claim 24, wherein urocortin is contained in a composition with bFGF.